far. To identify genes and pathways regulating vertebrate MZT, we have performed a unique phenotypic screen in Xenopus embryo. A number of potential MZT regulators have been identified. These include RNA binding proteins, protein kinase, epigenetic regulators, and signaling molecules. Our work thus builds up an important foundation for studying epigenetic regulation of gene expression during vertebrate MZT.

doi:10.1016/j.ydbio.2010.05.421

Program/Abstract # 259
Serine protease activation of the epidermal wound response in Drosophila
Rachel Patterson, William J. McGinnis
UCSD, La Jolla, CA 92093, USA

Following injury, breaches in the skin or cuticle are repaired by the epidermal wound response to restore barrier integrity. However, the manner by which nearby unwounded epidermal cells sense the wound and begin the process of repair is largely unknown. Here we show that treatment of embryonic epidermal cells with the serine protease trypsin can activate a global wound response. Part of the evidence for this is that wound enhancers from four different genes, originally identified by their abilities to be activated and localized around epidermal puncture wounds, are activated throughout the epidermis by serine protease treatment. The genes activated by this treatment include Dopa decarboxylase (Ddc) and tyrosine hydroxylase (ple), chitin synthase (kkv), and Misshapen (msn). Serine protease activation of the epidermal wound response can be activated by body cavity injection. This serine protease activation can also be effected by injection into the perivitelline space, which is not associated with a loss of epidermal integrity. Injections of the serine protease inhibitor aprotinin resulted in highly reduced expression levels of the wound response gene msn surrounding the wound site. Proteases from other families, such as the cysteine protease papain, do not activate the epidermal wound response as robustly. Serine protease treatment is likely to generate widespread activation of a wound response ligand, initiate a signaling pathway, and activate genes necessary for restoring epidermal integrity. We have used the trypsin-mediated wound response to screen Drosophila microarrays to determine the genomic response to epidermal wounding in late embryos/early larvae.

doi:10.1016/j.ydbio.2010.05.422

Program/Abstract # 260
Functional analysis of a UBX-responsive regulatory element
Bradley Hersh[a,b], Jamie L. Wood[b]
[a]*Dept. of Biology, Allegheny College, Meadville, PA, USA*
[b]*Dept. of Biol. Sci., Clemson University, Clemson, SC, USA*

Although Hox proteins play a major recognized role in establishing anterior–posterior pattern in developing animals, identification of direct target genes is challenging. Most Hox proteins can bind the DNA sequence TAAT, and co-factors, such as Extradenticle, can increase the DNA binding specificity. However, in many instances specific co-factors are not known, so the mechanisms for discriminating between target and not-target sequences are poorly characterized. Only a single conserved Ultrabithorax (UBX) binding site is necessary for the activation of a cis-regulatory element (CRE) for the CG13222 gene in the developing *Drosophila melanogaster* haltere. Here we have identified an additional sequence important for the activation of this gene through characterization of a minimal CRE and mutagenesis of specific sequences flanking the critical UBX site. Additionally, we have introduced homologous CRE sequences from several species of *Drosophila* into *D. melanogaster*. Changes in the expression pattern driven by the *D. ananassae* CRE suggest alterations in the cis-sequences regulating expression. In addition, the *D. pseudoobscura* CRE when introduced into *D. melanogaster* drives an expression that does not match the endogenous *D. pseudoobscura* pattern, suggesting changes in the trans-regulatory landscape between the two species. Therefore, at this single CRE, we are able to observe changes in both cis- and trans- that affect regulation of a UBX-target gene.

doi:10.1016/j.ydbio.2010.05.423

Program/Abstract # 261
Investigating the regulatory sequences of dpp required for negative feedback of dpp transcription
Maryanna Aldrich, Lorena Soares, Kristi Wharton
MCB Dept, Brown University, Providence, RI, USA

Patterning a uniform field of cells can be achieved by positional information provided by morphogen gradients. In the Drosophila wing disc, the BMP signaling pathway acts as a morphogen through a graded distribution of phosphorylated transducer, Mad. The pMad gradient, formed in response to the ligands *dpp* and *gbb*, directs distinct transcriptional responses of target genes to specify cell fates. As a morphogen system, it is vital that cells receive proper levels of signaling and that both generation and maintenance of the pMad gradient are highly regulated. Work in our lab has demonstrated that a negative feedback loop exists in the wing disc on *dpp* transcription. We believe that this serves to "fine tune" signaling in the event of altered activity levels. Expression of *dpp* reporters respond to BMP signaling, showing increased expression when signaling is reduced and decreased expression when signaling is increased. These experiments seek to identify the *dpp* enhancer sequences required for negative feedback. I have tested several reporters containing different regions of *dpp* enhancer sequence for their response to BMP signaling levels. I have also tested the requirement for binding of known *dpp* regulators by examining these reporters with mutated transcription factor binding sites. I have determined that BMP signaling regulates both the level and domain of *dpp* expression. In addition, feedback is not due solely to any one of the factors known to regulate the wing disc *dpp* expression that I have tested so far. Further experiments aim to identify the factors required for negative feedback of *dpp* and the mechanism by which this is achieved.

doi:10.1016/j.ydbio.2010.05.424

Program/Abstract # 262
Akirin links Twist transcription factor activity with the Brahma chromatin remodeling complex during embryogenesis
Scott J. Nowak[a], Hitoshi Aihara[b], Katie Gonzalez[a], Yutaka Nibu[b], Mary K. Baylies[a]
[a]*Dev. Bio., Sloan Kettering Inst., New York NY, USA*
[b]*Cell and Dev. Bio, Weill Cornell Medical College, New York NY, USA*

The activities of developmentally critical transcription factors (TFs) are regulated via interactions with accessory proteins. Such interactions either directly influence TF activity through binding and dimerization or indirectly promote gene activation by promoting a favorable chromatin environment for gene activation. Using a modified yeast two-hybrid screen, we identified *akirin*, a highly conserved nuclear protein, as a novel cofactor of the *Drosophila* muscle transcriptional regulator, Twist. Like *twist* hypomorphic